What is Claimed:

1. A method of inhibiting release of CD40 ligand, thromboxanes, or prostaglandin E2, or inhibiting CD40 ligand surface expression, by mammalian platelets, the method comprising:

contacting mammalian platelets with an effective amount of PPARγ, a PPARγ agonist, an RXR agonist, or a combination thereof, whereby said contacting inhibits release of CD40 ligand, thromboxanes, prostaglandin E2, or a combination thereof, and/or inhibits CD40 ligand surface expression by the mammalian platelets.

2. The method according to claim 1 wherein the mammalian platelets are human platelets.

3. The method according to claim 2 wherein the PPARγ is human PPARγ.

4. The method according to claim 1 wherein said contacting inhibits release of CD40 ligand by platelets.

5. The method according to claim 1 wherein said contacting inhibits release of thromboxanes by platelets.

6. The method according to claim 5 wherein the thromboxane is thromboxane B_2.

7. The method according to claim 1 wherein both the PPARγ agonist and the RXR agonist contact the mammalian platelet.

8. The method according to claim 1 wherein said contacting inhibits CD40 surface expression by platelets.

9. The method according to claim 1 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

10. The method according to claim 1 wherein the RXR agonist is 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

11. The method according to claim 1 wherein the mammalian platelets are *in vitro* or *ex vivo*.

12. The method according to claim 1 wherein the mammalian platelets are *in vivo*.

13. The method according to claim 12 further comprising:

administering PPARγ, the PPARγ agonist, the RXR agonist, or an inducer of a PPARγ agonist to a mammal in a manner that provides for said contacting.

14. The method according to claim 13 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

15. The method according to claim 13 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously, intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

16. The method according to claim 12 further comprising

administering a DNA molecule encoding PPARγ or an inducer of a PPARγ agonist to a mammal under conditions effective to cause transformation of one or more cells in a target tissue, thereby promoting expression of PPARγ or the inducer of a PPARγ agonist in the target tissue.

17. A method of inhibiting thrombosis, the method comprising:

contacting mammalian platelets with an effective amount of PPARγ, a PPARγ agonist, an RXR agonist, or a combination thereof, whereby said contacting inhibits formation of a thrombosis by the mammalian platelets.

18. The method according to claim 17 wherein the mammalian platelets are human platelets.

19. The method according to claim 18 wherein the PPARγ is human PPARγ.

20. The method according to claim 17 wherein both the PPARγ agonist and the RXR agonist contact the mammalian platelet.

21. The method according to claim 17 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

22. The method according to claim 17 wherein the RXR agonist is 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

23. The method according to claim 17 further comprising:

administering the PPARγ agonist, the RXR agonist, or an inducer of a PPARγ agonist to a mammal in a manner that provides for said contacting.

24. The method according to claim 23 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

25. The method according to claim 23 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously,

intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

26. The method according to claim 17 further comprising administering a DNA molecule encoding PPARγ or an inducer of a PPARγ agonist to a mammal under conditions effective to cause transformation of one or more cells in a target tissue, thereby promoting expression of PPARγ or the inducer of a PPARγ agonist in the target tissue in a manner effective to cause said contacting.

27. A method of treating or preventing a thrombotic condition or disorder, the method comprising:

contacting mammalian platelets, in an individual exhibiting symptoms of or predisposed to a thrombotic condition or disorder, with an effective amount of PPARγ, a PPARγ agonist, an RXR agonist, or a combination thereof, whereby said administering inhibits platelet activation to treat or prevent the thrombotic condition or disorder.

28. The method according to claim 27 wherein the mammalian platelets are human platelets and the individual is a human.

29. The method according to claim 28 wherein the PPARγ is human PPARγ.

30. The method according to claim 27 wherein both the PPARγ agonist and the RXR agonist contact the mammalian platelet.

31. The method according to claim 27 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

32. The method according to claim 27 wherein the RXR agonist is 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

33. The method according to claim 27 further comprising:

administering PPARγ, the PPARγ agonist, the RXR agonist, or an inducer of a PPARγ agonist to the individual in a manner that provides for said contacting.

34. The method according to claim 33 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

35. The method according to claim 33 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously, intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

36. The method according to claim 27 further comprising

administering a DNA molecule encoding PPARγ or an inducer of a PPARγ agonist to the individual under conditions effective to cause transformation of one or more cells in a target tissue, thereby promoting expression of PPARγ or the inducer of a PPARγ agonist in the target tissue in a manner effective to cause said contacting.

37. The method according to claim 27 wherein the thrombotic condition or disorder is selected from the group consisting of stroke, venous or arterial thrombosis, disseminated intravascular coagulation, myocardial infarction, pulmonary thrombo-embolism, and pulmonary hypertension.

38. A method of improving the quality of a blood product, the method comprising:

providing PPARγ, a PPARγ agonist, an RXR agonist, an inducer of a PPARγ agonist, or a combination thereof; and

introducing PPARγ, the PPARγ agonist, the RXR agonist, the inducer of a PPARγ agonist, or the combination thereof, to a blood product, wherein the PPARγ agonist, the RXR agonist, the inducer of a PPARγ agonist, or the combination thereof inhibits clotting or activation of platelets in the blood product and thereby improves the quality thereof.

39. The method according to claim 38 wherein the blood product is selected from the group consisting of whole blood, plasma, concentrated platelets, or a white blood cell product.

40. The method according to claim 38 wherein the blood product is a mammalian blood product.

41. The method according to claim 40 wherein the mammalian blood product is a human blood product.

42. The method according to claim 41 wherein the PPARγ is human PPARγ.

43. The method according to claim 38 wherein said introducing is carried out prior to storage of the blood product.

44. The method according to claim 38 wherein the blood product is whole blood and said introducing comprises:

collecting whole blood from a patient into a receptacle comprising the PPARγ agonist.

45. The method according to claim 38 wherein the blood product is plasma or concentrated platelets and said introducing comprises:

collecting whole blood from a patient;

separating the plasma or concentrated platelets from the whole blood; and

combining the PPARγ agonist, the RXR agonist, the inducer of a PPARγ agonist, or the combination thereof, with the plasma or concentrated platelets.

46. The method according to claim 38 wherein the blood product is plasma or concentrated platelets and said introducing comprises:

collecting whole blood from a patient;

combining the PPARγ agonist, the RXR agonist, the inducer of a PPARγ agonist, or the combination thereof, with the whole blood to form a treated mixture; and

separating the plasma or concentrated platelets from the treated mixture.

47. The method according to claim 38 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

48. The method according to claim 38 wherein the RXR agonist is selected from the group of 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

49. The method according to claim 38 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

50. A stored blood product comprising:

a blood product that contains platelets and

an amount of PPARγ, a PPARγ agonist, an RXR agonist, an inducer of a PPARγ agonist, or a combination thereof that is effective to inhibit platelet activation.

51. The stored blood product according to claim 50 further comprising an anticoagulant.

52. The stored blood product according to claim 50 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

53. The stored blood product according to claim 50 wherein the RXR agonist is selected from the group of 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

54. The stored blood product according to claim 50 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

55. The stored blood product according to claim 50 wherein the blood product is whole blood, plasma, concentrated platelets, or a white blood cell product.

56. A method of inhibiting platelet aggregation comprising:

contacting mammalian platelets with an effective amount of PPARγ, a PPARγ agonist, an RXR agonist, or a combination thereof, whereby said contacting inhibits aggregation of the mammalian platelets.

57. The method according to claim 56 wherein the mammalian platelets are human platelets.

58. The method according to claim 57 wherein the PPARγ is human PPARγ.

59. The method according to claim 56 wherein both the PPARγ agonist and the RXR agonist contact the mammalian platelet.

60. The method according to claim 56 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

61. The method according to claim 56 wherein the RXR agonist is 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

62. The method according to claim 56 further comprising:

administering the PPARγ agonist, the RXR agonist, or an inducer of a PPARγ agonist to a mammal in a manner that provides for said contacting.

63. The method according to claim 62 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

64. The method according to claim 62 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously, intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

65. The method according to claim 56 further comprising

administering a DNA molecule encoding PPARγ or an inducer of a PPARγ agonist to a mammal under conditions effective to cause transformation of one or more cells in a target tissue, thereby promoting expression of PPARγ or the

inducer of a PPARγ agonist in the target tissue in a manner effective to cause said contacting.

66. A method of treating or preventing a CD40 ligand-mediated or thromboxane-mediated condition, the method comprising:

contacting platelets, in an individual exhibiting or predisposed to a CD40 ligand-mediated or thromboxane-mediated condition, with PPARγ, a PPARγ agonist, an RXR agonist, an inducer of a PPARγ agonist, or a combination thereof, whereby said contacting inhibits the release of CD40 ligand and/or thromboxane by platelets, thereby treating or preventing the CD40 ligand-mediated or thromboxane-mediated condition.

67. The method according to claim 66 wherein the PPARγ is human PPARγ.

68. The method according to claim 66 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

69. The method according to claim 66 wherein the RXR agonist is selected from the group of 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

70. The method according to claim 66 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

71. The method according to claim 66 wherein the individual is human.

72. The method according to claim 66 wherein the CD40 ligand-mediated or thromboxane-mediated condition is selected from the group consisting of diabetes, atherosclerosis, induced multiple sclerosis, asthma, venous or arterial thrombosis, pulmonary fibrosis, systemic lupus erythematosus, renal fibrosis, hepatic

cirrhosis, cerebral gliosis, disseminated intravascular coagulation, myocardial infarction, pulmonary thrombo-embolism, and pulmonary hypertension.

73. The method according to claim 66 wherein said contacting is carried out by administering PPARγ, the PPARγ agonist, the RXR agonist, the inducer of a PPARγ agonist, or the combination thereof to the individual in a manner effective to cause said contacting.

74. The method according to claim 73 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously, intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

75. The method according to claim 52 wherein said contacting is carried out by administering to the patient a DNA molecule encoding PPARγ or the inducer of a PPARγ agonist, said administering being carried out under conditions effective to cause transformation of one or more cells in a target tissue in a manner effective to cause said contacting.

76. The method according to claim 75 wherein the DNA molecule is in an expression vector or present as naked DNA.

77. The method according to claim 76 wherein the expression vector is an adenoviral vector or adenoviral associated vector

78. A method of assessing the activity of a compound as a PPARγ agonist, the method comprising:

combining a compound with both platelets and a platelet activator;

determining the level of CD40 ligand or thromboxane released from the platelets; and

comparing the level of CD40 ligand or thromboxane released from the platelets to the level of CD40 ligand or thromboxane released from a standard,

wherein deviation from the standard, or absence thereof, indicates activity of the compound as a PPARγ agonist.

79. The method according to claim 78 wherein said determining comprises:

providing an antibody or binding portion thereof recognizing CD40 ligand or an antibody or binding portion thereof recognizing the thromboxane;

contacting a sample from the combination with the antibody or binding portion thereof; and

detecting any reaction which indicates that CD40 ligand or thromboxane is present in the sample using an assay system.

80. The method according to claim 79 wherein the assay system is selected from a group consisting of enzyme-linked immunoabsorbent assay, radioimmunoassay, gel diffusion precipitin reaction assay, immunodiffusion assay, agglutination assay, fluorescent immunoassay, protein A immunoassay, immunoelectrophoresis assay, Western blot, immunodotblot, and immunoslotblot.

81. The method according to claim 78 wherein the standard comprises platelets in the absence of the activator, and said comparing assesses the absence of deviation between the combination and the standard.

82. The method according to claim 78 wherein the standard comprises platelets in the presence of the platelet activator, and a known PPARγ agonist, and said comparing assesses the deviation between the combination and the standard.

83. The method according to claim 78 wherein the platelet activator is thrombin, epinephrine, collagen, or ADP.

84. A method of diagnosing a CD40 ligand-mediated condition comprising:

obtaining a patient sample and

determining the level of PPARγ in the patient sample, wherein a reduced PPARγ level indicates the presence of a CD40 ligand-mediated condition.

85. The method according to claim 84 wherein said determining comprises:

providing an antibody or binding portion thereof recognizing PPARγ;

contacting the sample with the antibody or binding portion thereof; and

detecting any reaction which indicates that PPARγ is present in the sample using an assay system.

86. The method according to claim 85 wherein the assay system is selected from a group consisting of enzyme-linked immunoabsorbent assay, radioimmunoassay, gel diffusion precipitin reaction assay, immunodiffusion assay, agglutination assay, fluorescent immunoassay, protein A immunoassay, immunoelectrophoresis assay, Western blot, immunodotblot, and immunoslotblot.

87. The method according to claim 84 wherein the CD40 ligand-mediated condition is selected from the group consisting of diabetes, atherosclerosis, induced multiple sclerosis, asthma, venous or arterial thrombosis, pulmonary fibrosis, systemic lupus erythematosus, renal fibrosis, hepatic cirrhosis, cerebral gliosis, disseminated intravascular coagulation, myocardial infarction, pulmonary thrombo-embolism, and pulmonary hypertension.

88. The method according to claim 84 wherein the patient sample is selected from the group of blood, plasma, tissue washings, lung lavage, eye fluids, saliva, joint fluid, peritoneal fluid, stool, semen, gastric fluids, and thoracic fluids.

89. A method of assessing the efficacy of a PPARγ agonist therapy, the method comprising:

obtaining a patient sample, the patient having been previously administered a PPARγ agonist or an inducer of a PPARγ agonist for treating a medical condition or disorder; and

determining the level of PPARγ in the patient sample, wherein an elevated PPARγ level, relative to a baseline PPARγ level for the patient prior to said administration, indicates the efficacy of the PPARγ agonist therapy.

90. The method according to claim 89 wherein said determining comprises:

providing an antibody or binding portion thereof recognizing PPARγ;

contacting the sample with the antibody or binding portion thereof; and

detecting any reaction which indicates that PPARγ is present in the sample using an assay system.

91. The method according to claim 90 wherein the assay system is selected from a group consisting of enzyme-linked immunoabsorbent assay, radioimmunoassay, gel diffusion precipitin reaction assay, immunodiffusion assay, agglutination assay, fluorescent immunoassay, protein A immunoassay, immunoelectrophoresis assay, Western blot, immunodotblot, and immunoslotblot.

92. A method of modifying megakaryocytes comprising:

exposing a megakaryocyte to PPARγ, a PPARγ agonist, an RXR agonist, an inducer of a PPARγ agonist, or a combination thereof, whereby said exposing phenotypically modifies the megakaryocyte to produce daughter platelets that minimize pro-inflammatory and/or pro-thrombotic responses by the platelets.

93. The method according to claim 92 wherein the PPARγ is human PPARγ.

94. The method according to claim 92 wherein the PPARγ agonist is selected from the group consisting of cyclopentenone class prostaglandins, lysophosphatidic acid or its derivatives, thiazolidinediones, glitazones, tyrosine-derived agonists, indole-derived agonists, and combinations thereof.

95. The method according to claim 92 wherein the RXR agonist is selected from the group of 9-*cis*-retinoic acid, *trans*-retinoic acid, synthetic RXR agonists, and combinations thereof.

96. The method according to claim 92 wherein the inducer of a PPARγ agonist is decorin or fragments thereof, or an enzyme that catalyzes formation of prostaglandin D_2 precursor.

97. The method according to claim 92 wherein the megakaryocyte is a human megakaryocyte.

98. A method of treating or preventing a CD40 ligand-mediated or thromboxane-mediated condition, the method comprising:

treating a patient exhibiting or predisposed to a CD40 ligand-mediated condition with recombinant PPARγ, whereby said treating inhibits the release of CD40 ligand and/or thromboxane by platelets, thereby treating or preventing the CD40 ligand-mediated or thromboxane-mediated condition.

99. The method according to claim 98 wherein the patient is human.

100. The method according to claim 98 wherein the CD40 ligand-mediated condition is selected from the group consisting of diabetes, atherosclerosis, induced multiple sclerosis, asthma, venous or arterial thrombosis, pulmonary fibrosis, systemic lupus erythematosus, renal fibrosis, hepatic cirrhosis, cerebral gliosis, disseminated intravascular coagulation, myocardial infarction, pulmonary thrombo-embolism, and pulmonary hypertension.

101. The method according to claim 98 wherein said treating is carried out by administering the recombinant PPARγ to the patient.

102. The method according to claim 101 wherein said administering is carried out via topical application, intranasal instillation, inhalation, intravenous injection, intra-arterial injection, intramuscular injection, application to a wound site, application to a surgical site, intracavitary injection, by suppository, subcutaneously, intradermally, transcutaneously, by nebulization, intraplurally, intraperitoneally, intraventricularly, intra-articularly, intra-aurally, intraocularly, or intraspinally.

103. The method according to claim 98 wherein said treating is carried out by administering to the patient a DNA molecule encoding the recombinant PPARγ, said administering being carried out under conditions effective to cause transformation of one or more cells in a target tissue, thereby promoting expression of the PPARγ in the target tissue.

104. The method according to claim 103 wherein the DNA molecule is in an expression vector or is naked DNA.

105. The method according to claim 103 wherein the expression vector is an adenoviral vector or an adeno-associated viral vector.